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AD-000 188

UNCLASSIFIED/LIMITED DTIC AD 188

Report No. 151
Chemical Corps Medical Laboratories
Research Report

CASE REPORT OF A SEVERE HUMAN POISONING BY CB

Case Report from Dugway Proving Ground of Severe Human Poisoning by GB

by

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December 1952

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Medical Laboratories Research Report No. 151

Case Report of a Severe Human Poisoning by GB

I. INTRODUCTION

On 7 November 1952, during the field testing of GB-filled spray tanks at Dugway Proving Ground, Tooele, Utah, a case of severe human poisoning by the agent occurred. The casualty failed to observe elementary safety precautions and was poisoned by the respiratory route. The patient, a 32 year old white male, represents the most severe "G" casualty observed up to this time. However, he has made an apparently complete recovery.

The recording of observations and treatment made during the period immediately following exposure were, of course, secondary to efforts to keep the patient alive. Only a medical aid man was available for the care of this patient during the first 20 minutes of his illness. Nevertheless, it is felt that this is a reasonably accurate description of the signs and symptoms which resulted from this exposure.

II. CIRCUMSTANCES OF EXPOSURE

According to the test plan a jet aircraft equipped with wing tanks, each containing 100 gallons of GB, sprayed the agent over a target site. Because of the malfunctioning of the tanks they still contained approximately 90 gallons of GB each which jettisoned in an isolated area. They were dropped at 0829 hours from an estimated height of 2,000 feet and burst on contact with the ground, spreading a high concentration of agent over an area of 38,000 square feet. The contaminated area could be easily identified, since the liquid was colored with a red dye.

The basic terrain of the contaminated region was flat, open desert with a salt crust, resistant to fluid absorption and covered with very sparse desert shrubbery. The day was clear and cool with an air temperature of 42° F at the time of exposure. The winds were from approximately the north and averaged three to four mph.

An inspection crew, consisting of two safety personnel, a decontamination crew of two, a medical officer (b) (6), and aid man, proceeded to this area. Thirty minutes before this group arrived at the contaminated region (approximately three miles to the southeast) they all donned their gas masks with the exception of (b) (6), who ignored the advice of those present. They arrived at 1140 hours, three hours after the tanks had been released, and parked the ambulance 200 feet upwind. (b) (6), still unmasked and without protective clothing,

promptly left the ambulance and slowly walked to within 10 feet of the agent-filled crater made by the tank when it fell. Standing northeast of the contaminated ground, he was upwind.

III. OBJECTIVE MANIFESTATIONS OF POISONING

(b) (6) was observed to walk toward the area of liquid contamination in a normal manner. Within 10 seconds he turned, clutched his chest and started toward the ambulance at a fast walk. He called frantically for his gas mask. His gait soon became stumbling, and as he staggered one arm extended and flexed in a jerky manner. He collapsed upon reaching the ambulance. After his gas mask was applied, he was immediately given atropine by deep intramuscular injection in the anterior thigh (estimated dose 1 mg) and put into the ambulance to be evacuated to the Post Hospital. At this time his respirations were regular but stridulous. The inspiratory phase of respiration was accompanied by a high-pitched "screeching" sound, and low-pitched "gargles" on expiration. He was noted to have convulsive jerks recurring at five second intervals and lasting for about one second. These motions consisted of hyperextension of the spine and legs and rapid, forceful extension of the abducted arms above the head. This convulsive pattern lasted for 60 seconds and was replaced by flaccid paralysis. Respiration became irregular with periods of apnea alternating with three to four deep breaths; stridor continued.

Two minutes later (approximately five minutes after exposure) respirations were markedly reduced with only an occasional gasp. The mask was removed and the excessive secretions were wiped from the exterior part of the mouth and the lips with a piece of gauze before starting resuscitation with an Emerson portable respirator. There was no evident obstruction as the respirator functioned normally, but secretions rapidly reaccumulated. The patient was given 2 mg atropine by deep intramuscular injection in the left anterior thigh. At this time he had complete flaccid paralysis which included the respiratory muscles. The pupils were pinpoint and the eyes were open, fixed, and staring in a central position. No arterial pulse could be detected by the aid man.

(b) (6) condition remained unchanged until 10 minutes later when the resuscitator began to cycle rapidly, indicating an obstruction. Deep cyanosis soon developed.

It was at this time, approximately 25 minutes after the exposure that a medical officer intercepted the ambulance on its way to the hospital. Breathing was characterized by coarse, bubbling, inspiratory and expiratory growls of low-pitch. Aspiration with the suction device of the Emerson portable resuscitator was ineffectual as the suction created was not

strong enough to remove the thick mucoid secretions. As much of these secretions as possible was removed with a piece of gauze, an airway inserted, and the oxygen mask reapplied. The rapid cycling still indicated an obstruction so the instrument was switched to "inhalation" whereby a rubber bag could be filled with oxygen and forced into the victim by manual pressure on the bag. The aid man completed the respiratory cycle by pressure on the thorax to produce forced exhalation. There was some improvement in color but cyanosis persisted. At this time (b)(6) had complete flaccid paralysis. The skin was deep blue and dry. There was moderate conjunctival injection; the conjunctival sacs were dry and filled with dust and debris. The lids were still open, the eyes were fixed in a central position, and the pupils were pinpoint. There was a moderate accumulation of thick mucous in the mouth and pharynx. The radial pulse was weak but regular at a rate of 160/min. Two milligrams atropine were given into the right anterior thigh and repeated in five minutes.

Thirty-five minutes after exposure, J.A. was admitted to the U.S. Army hospital.

IV. HOSPITAL COURSE

The patient was admitted to the hospital at 1230 hours and immediately placed in an "iron lung" type resuscitator. Preparations were made to do a tracheotomy but the cyanosis disappeared as soon as the secretions were removed with an efficient aspirator and a better airway inserted. Oxygen was continued via a nasal catheter. The face was an ashen gray color and the skin was dry. The blood pressure was 150/105 and pulse 140. There was a complete flaccid paralysis associated with a loss of the deep tendon reflexes and there was no reaction to painful stimuli. The pupils were pinpoint and the conjunctivae were hyperemic, dry and covered with dust. The nasal turbinates were swollen and congested. A moderate amount of thick, gray mucous was present in the nasal passageways and the oropharynx was filled with similar viscid secretions. These secretions were aspirated with difficulty and continued to reaccumulate throughout the remainder of that day and evening.

By 75 minutes after exposure, diaphragmatic breathing of a sporadic nature was noted and this interfered with adequate air exchange in the mechanical lung. Deep tendon reflexes returned although the patient was still flaccid and unconscious. Positive pressure was discontinued, and with only rhythmic negative pressure in the iron lung, the patient stopped resisting the respiratory pattern imposed by the machine. Throughout this period repeated aspiration of pharynx and larynx was necessary.

One hundred minutes after exposure, and after 60 minutes in the iron lung, movements of the intercostal muscles

began. The use of the mechanical lung was temporarily discontinued but spontaneous breathing proved to be erratic. It consisted of three to four deep gasping breaths alternating with 20- to 30-second periods of apnea. Adequate auscultation of the heart and lungs was possible for the first time. The inspiratory phase was longer than the expiratoty, and no wheezes or rales could be heard. The heart sounds were normal and the rhythm was regular. The blood pressure was then 120/80 and the radial pulse 120. Because of the weak, irregular breathing artificial respiration was continued.

One hundred and thirty-five minutes after exposure, spontaneous motion of the arms was noted. Because the patient's hands involuntarily grasped at his throat, it was necessary to forcibly restrain his upper extremities. The deep tendon reflexes were normal, and there was slight withdraw from painful stimuli. The pupils remained pinpoint and the eyes fixed in a forward stare. The blood pressure was 110/80, pulse 94, regular and full. The heart and lungs were clear to auscultation. Respiration was still irregular with long periods of apnea between deep breaths, and use of the iron lung was continued. Color remained good and an eventual recovery was judged probable.

At 165 minutes (b) (6). began to speak in a halting but intelligible manner. There was now voluntary and controlled movement of all extremities. He was removed from the respirator, and respiration was maintained spontaneously at a regular rate of 24 per minute. The schedule of atropine at about 15 minute intervals was stopped. The blood pressure and pulse remained unchanged. Repeated aspiration of accumulated mucous was still necessary. The patient appeared alert and oriented, although he complained of severe malaise. Fasciculations of the masseter muscle were noted and prompted the administration of a final 2 mg dose of atropine at 1500 hours.

Thereafter, the patient rested well and complained only of photophobia, frontal and retrobulbar headache, and mild nausea. His blood pressure remained at 110-120/65-80 and his pulse rate at 75-85. Respirations continued at a regular rate of 18-22 per minute. Nasal oxygen was discontinued. A physical examination at this time was normal except for moderate weakness, pupillary constriction and nasal congestion. He was alert and oriented, although slightly lethargic. The heart, lungs, breath sounds, superficial and deep reflexes were all essentially normal by the usual clinical standards. Exteroception and proprioception were grossly normal. Involuntary urination and defecation did not occur at any time.

V. CONVALESCENCE

By three and one-half hours after exposure, it was apparent that (b) (6) had essentially recovered from his poisoning. Throughout the evening and night of 7 November, he complained of a dry mouth and anorexia. Whenever oral fluids were taken, he promptly vomited bile-stained, thick mucoid material. There were no abdominal cramps, diarrhea, constipation, or urinary symptoms. Urine was voided for the first time that evening. His temperature and other vital signs remained normal.

The pupils were constricted until the fifth hospital day when they first reacted to light and accommodation; by the ninth hospital day they were apparently normal. The patient first became ambulatory the morning after exposure. No objective difficulty in locomotion was demonstrable, but for the next three days (b) (6) elected to spend most of his time in bed. By the second afternoon fluids were consistently retained, and on the third morning a regular diet was readily tolerated. Thereafter, his appetite was good. He was withdrawn and indifferent, but this undemonstrative behavior was consistent with his pre-exposure personality. No intellectual impairment was evident.

(b) (6) was evacuated by aircraft to the Oak Knoll Naval Hospital, Oakland, California, on 27 November 1952.

VI. LABORATORY OBSERVATIONS

Hematological data were normal except for a WBC of 15,000 per cm (with a slight shift to the left) on the afternoon of exposure. A routine urinalysis was unremarkable.

Blood cholinesterase activity was measured by Dr. (b) (6) Chief of the Enzymology Section of the CW Division. The following data were taken from a CW interim report by the CW Division Chief (Dr. (b) (6). [redacted]. As obtained by a manometric method, the activities are expressed in micromoles of acetylcholine chloride hydrolyzed per hour per ml of packed red blood cells or per ml of heparinized plasma.

Hours after Exposure	Plasma	RBC
2.0	8.8	0
3.5	14.0	0
5.8	19.1	0
21.8	35.2	0
30.0	42.0	12.2
46.0	49.2	16.4
68.0	64.5	20.9
134.0	90.0	15.0
227.0	100.0	40.0
410.0	145.0	66.0

From a sample of pooled normal human plasma, the value obtained was 146 micromoles of acetylcholine hydrolyzed per hour per ml of plasma. Samples from five normal individuals averaged 400 micromoles of acetylcholine hydrolyzed per hour per ml of packed red blood cells.

VII. SUBJECTIVE REACTIONS OF THE PATIENT

(b) (6) recalled no symptoms until he stopped near a shallow ground crater containing liquid agent. He detected a faint odor which he describes as resembling gasoline fumes. Within 10 seconds he felt "giddy," and this sudden sensation of faintness (without vertigo) prompted him to start toward the ambulance. He remembered nothing from this moment until his recovery in the hospital. In the few seconds before loss of consciousness, he was aware of no dyspnea, constrictive feeling of the chest, rhinorrhea, or muscle twitching.

After gaining consciousness in the hospital, his prominent symptoms were recurrent nausea, photophobia, and headache. The headache was frontal and retrobulbar, but it was not severe. Nausea was noticeable only whenever the patient attempted to drink fluids, at which point he invariably vomited. No abdominal pain or urinary symptoms were admitted. Both the nausea and headache subsided the next morning, while anorexia persisted throughout the second day. Fluids were well retained by the second afternoon, and on the third morning a regular diet was taken without distress.

Photophobia gradually lessened but was annoying for two to three days. On the third morning, he managed to read Sunday comics without ocular pain, but this required intense concentration to avoid blurring. Throughout this period his distance vision was definitely blurred and was not subjectively normal until about the 7th hospital day.

Throughout his hospitalization he experienced no insomnia, except on the first night when sleep was interrupted by coughing and retching. No disturbing dreams were acknowledged. The patient was aware of no change in his sexual libido during his stay in the hospital. No muscular weakness, difficulties in gait, or other neuromuscular symptoms were experienced, even when the patient first left his bed on the morning after exposure.

VIII. TREATMENT

First aid in the field consisted of intramuscular atropine, aspiration of the oropharynx, the introduction of an oropharyngeal airway, and artificial respiration, as outlined previously.

When admitted to the hospital, (b) (6). was placed in an iron lung of the Drinker type, by which respiration was sustained for two hours. Two milligrams of atropine were given into the anterior thigh and preparations for a tracheotomy were made, because rattling sounds were still present whenever air was moved through the pharynx and trachea. However, with adequate aspiration of accumulated secretions, and the use of a large, hard rubber oropharyngeal airway, the obstruction was cleared and the abnormal sounds disappeared. Oxygen was administered by nasal catheter. Over the next two hours, (b) (6) received 2 mg atropine intramuscularly approximately every 15 minutes. The total dose given over a period of three hours was 19 mg. The last dose of intramuscular atropine was administered at 1500 hours; atropine was not given orally at any time. No tracheotomy was performed.

Once an adequate air exchange was established and before the disappearance of coma, the patient's eyes were irrigated with saline to remove dust. Castor oil was instilled in the conjunctival sacs, and the eyes were held closed with a light bandage. Procaine penicillin (300,000 units intramuscularly daily) was administered prophylactically for three days, beginning on the day of exposure. No other drugs or therapeutic measures were employed.

IX. DISCUSSION

The medical corpsman and safety officer were in essential agreement in their observations of (b) (6) reactions

during the immediate post-exposure period. Because of their vivid reporting, considerable confidence is placed upon the clinical picture during this period. Being the only medical officer at this location, J.A. was responsible for the emergency treatment of test casualties. As a result of his poisoning, it was 25 minutes before an examination by a physician was possible. Because of the rapidly changing clinical picture during this initial post-exposure period, certain details of (b) (6) reactions are lacking. Although it appears evident that he received a high dose of GB, quantitative measurements of vapor concentration were not obtained.

In interpreting the patient's intoxication, his rapid deterioration can be arbitrarily divided into four phases. The initial reaction, beginning within seconds after his arrival at the contaminated area, suggests cardiovascular collapse, characterized by giddiness, staggering, "blacking out", and a weak or absent radial pulse. Within a few moments, a convulsive phase intervened. This unusual convulsive pattern was accompanied by what is interpreted to have been laryngeal spasm, and the combination quickly led to severe anoxia. Perhaps because of anoxia, the laryngeal spasm soon subsided. Neuromuscular paralysis, whether of peripheral or central origin, erased the convulsive manifestations, but now because of respiratory paralysis anoxemia was intensified. In the initial phase of flaccid paralysis, artificial respiration with the Emerson resuscitator seemed to have been adequate and was probably life-saving. In a fourth phase, however, paralysis was complicated by the accumulation of nasal, pharyngeal, and tracheal secretions, which almost nullified all efforts to establish air exchange. No convincing evidence of bronchiolar spasm or pulmonary edema occurred. The major problem in the emergency treatment proved to be the removal of these secretions.

(b) (6) denied any history of allergic disturbances but he had long experienced a postnasal discharge every morning, a phenomenon which he ascribed to smoking cigarettes. It has been proposed that this disturbance is often evidence of physiological hyperactivity in the nasal secretory cells. If this concept is correct, such reactors might experience excessive difficulty from secretions during intoxication with anticholinesterase agents.

The rapidity of the patient's recovery in the hospital was spectacular. From the moment of adequate aspiration of pharynx and larynx, his recovery in the Drinker respirator was progressive. Even after artificial respiration and the administration of atropine were discontinued, no transient relapse in his clinical status was noted. In several respects his late symptoms were as mild and temporary as those seen in patients with apparently minimal exposures. For example, he experienced no abdominal pain, diarrhea, or urinary difficulty.

He complained of minor retrobulbar pain for only two days, but there was no appreciable tenderness from mild pressure on the eyeballs. Since distant objects remained blurred several days after near vision was adequate, ciliary spasm is presumed to have existed for about a week. Miosis disappeared within nine days.

The patient's rapid recovery is ascribed in large measure to the prompt and intense administration of 2 mg doses of atropine sulfate, totalling 19 mg over a period of three hours. This high dose may also have minimized many of the non-lethal but distressing sequelae which have been seen in milder casualties who were not treated. The patient's high tolerance to atropine was indicated by the absence of any definitive evidence of atropine intoxication. However, no atropine was required after the first three and one-half hours. This observation implies an unexpectedly long duration to the protection afforded by atropine, an effect which is possibly attributable to the unusually large dose. Although all doses were here given by intramuscular injection, it might have been advantageous to have injected the initial doses intravenously. In this patient, successful atropinization was accomplished by continued intramuscular administration at 10 to 20 minute intervals until voluntary and controlled movements of the extremities were noted and spontaneous respiration was regular.

X. SUMMARY

A severe human casualty from GB vapor was observed at Dugway Proving Ground, Tooele, Utah. The circumstances of exposure, the clinical signs and symptoms, the treatment, and the convalescence are here described. Serial observations of the red blood cell and plasma cholinesterase levels are included. Artificial respiration and treatment with large doses of atropine resulted in an apparently complete recovery.

XI. RECOMMENDATIONS

On the basis of this single case, the following recommendations are tentatively offered.

1. In severe GB poisoning the prompt administration of atropine should be started by intravenous injection.

2. Intramuscular injection of 2 mg doses of atropine should be continued at 10 to 20 minute intervals until breathing is maintained spontaneously.

3. First aid equipment should include:

a. Means for adequate aspiration of the oropharyngeal secretions.

b. A mechanical resuscitator.

c. Equipment for endotracheal intubation.

d. A tracheotomy set.